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Mineralizing Angiopathy causing basal ganglia infarct in infant due to minor trauma: A case report

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Abstract
Introduction: Basal ganglia infarct due any minor trauma in infants is a rare but definite clinico-radiological entity that is been termed as Mineralizing Angiopathy of Infancy. Healthy infants with no previously known systemic illness presents with hemiparesis and facial paresis following a minor injury. Supporting clinical findings on Neuroimaging it reveals non-hemorrhagic focal infarct in basal ganglia.
Case report: We report a case of a 2-year old female child presenting with left-sided hemiparesis following trauma. Neuroimaging of brain showed Right Basal Ganglia Infarct due to Mineralizing Angiopathy.
Conclusion: This case report emphasizes that Mineralizing Angiopathy should be included in the differential diagnosis in any infant who presents with acute infantile hemiplegia following minor trauma. The neurological outcome is usually good in most children.

Keywords: mineralizing angiopathy**,** basal ganglia infarct**,** minor trauma**,** lenticulostriate vasculopathy**,** capsulo-ganglionic lesion

Introduction

A cerebral vascular accident (CVA) or infarct occurring in children is rare disorder. Its combined incidence of ischemic and haemorrhagic paediatric CVA ranges from 1.2 to 13 cases per 100,000 children below 18 years of age [1]. Pediatrics infarct has a mortality rate of 0.6 per 100,000 deaths per year [2]. Mortality is high in such infants, but most of the surviving patients develops neurological and cognitive impairment and some develops epilepsy [3].

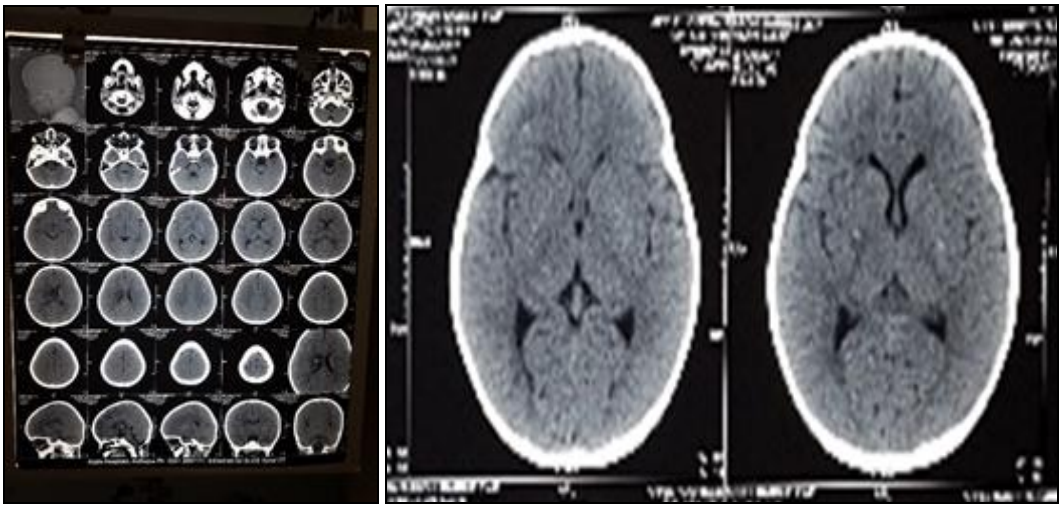
Basal ganglia infarct is considered rare, occurring in less than 2% of all paediatric CVA. Its occurrence in infants following trivial trauma is known and previously reported; however, its co-existence with mineralizing angiopathy has been recently established [4, 5, 6].

These children presents with hemiparesis with facial paresis and show small non-haemorrhagic infarct in basal ganglia on CT and/or MRI. It has characteristic presentation and evolution of symptoms. Previously healthy infants of age 6-24 months are usually affected, sudden onset with rapid progression of neurological deficit following a trivial trauma, affected side shows dystonia between 2 to 4 days which subsides within 24- 48 hours, bilateral basal ganglia calcification along with mineralization are seen on CT images. Short and long-term neurodevelopment is good with good prognosis except in infarct occurring in recurrent stroke [7].

Case report

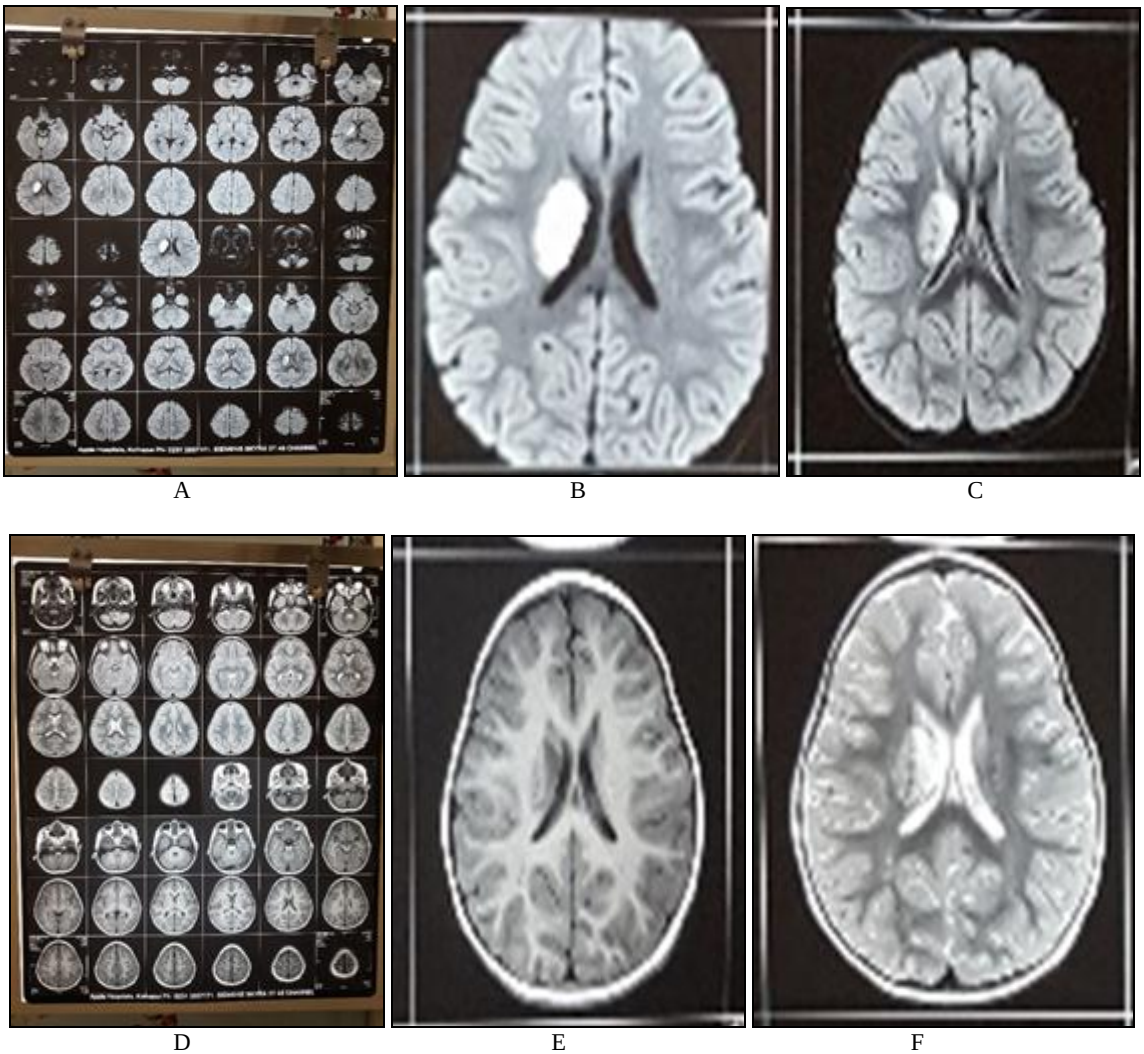
A 2-year old female child presented with sudden onset weakness of left upper and lower limbs following road traffic accident 1 day back. No history of loss of consciousness, seizures, bleeding, vomiting and fever. Birth history was normal. No history of any congenital heart disease. Milestones achieved were normal for age. Parents gave a history of non-consanguineous marriage.

On admission child was vitally stable. On CNS examination revealed, left-sided hemiparesis, decreased tone in left upper and lower limbs with brisk deep tendon reflexes and left plantar reflex was extensor. Sensory examination and examination of cranial nerves were normal. Results of laboratory investigations included hemoglobin concentration of 9.6 g/L, hematocrit value of 29.8%, white blood cell count of 9800/mm3 with 52% neutrophils, 40% lymphocytes, 5% eosinophil, 3% monocytes and the platelet count was 5.43 lac/mm3. Serum biochemical values, including C-reactive protein, electrolytes were in the normal range. Septic workup was negative. She was then further investigated with non-enhanced computed tomography (CT) and magnetic resonance imaging (MRI) of the brain demonstrated in figures below.



A B

Fig 2 A, B: CT scan of the brain: Bilateral basal ganglia Calcification. Hypodense area with Right Capsulo-Ganglionic Lesion was noted.



A B C

D E F

Fig 3 A,B,C,D,E,F: MRI scan of the brain revealed Axial Diffusion weighted image showing restriction with Right Capsulo Ganglionic lesion, T1 weighted image showing hyperintense area, T2- weighted images appearing hyper diffuse restriction. These findings were suggestive of the acute non-hemorrhagic infarct. MR angiography was normal.

In further more investigations 2D echocardiography was normal. The Antiphospholipid antibody, homocystenine and protein C and S levels were within normal limits.

The clinico-radiological presentation was consistent with

mineralizing angiopathy causing acute basal ganglia infarct.
The patient was treated with Injectable low molecular weight heparin (LMWH) 1mg/kg/day by subcutaneous route for 5 days and then shifted to oral 5mg/kg/day of aspirin for 7 days and physical rehabilitation. Following 5 days of treatment, improvement in power was noted with an ability walk with support and after 15 days to grasp an object with the left hand.

Discussion

Basal ganglia infarct following minor trauma is considered rare entity, with incidence less than 2% of all peadiatric ischemic stroke below the age of 2 years. Pathology behind Clinical manifestations and its radiologic findings are still unexplained [4]. The proposed mechanisms include transient arterial spasm, mechanical disruption of the flow in the perforating arteries, and intimal trauma with subsequent thrombosis. On neuroimaging, hyperdense foci were seen within the area of infarct which was diagnosed as thrombus or hemorrhage [8]. A detailed history of any traumatic events preceding the stroke must be recorded. Further, as the pediatric strokes have multifactorial etiology, work up for other causes should also be done.
Yang *et al.* showed basal ganglia calcifications in 10 out of 16 infants with stroke following minor trauma. These were likely to represent linear vascular mineralization not documented in this series. Pathophysiology of this vascular mineralization is however, unclear.
Yang *et al.* documented correlation between basal ganglia infarction following minor trauma and serological test for viral infective causes like cytomegalovirus (CMV), echovirus, Epstein-Barr virus (EBV) and mycoplasma [6]. Seropositivity of CMV was co-related with basal ganglia mineralization. Other infections were not associated independently with basal ganglia mineralization and were seen only in conjunction with CMV infection. Further research is required as it is not clear whether acute CMV infection causes mineralization.
Recently Ligappa *et al.* gave a distinct clinico-radiological entity for mineralizing angiopathy. Classically described in previously healthy infants below the age of 2 years having basal ganglia infarct following minor trauma can be a predisposing factor for thrombosis [7]. Even though the mineralization persist in the vessels after minor trauma, infants remains asymptomatic beyond 2-3 years of age, and their neurodevelopmental outcome is also good.
According to 2007 National Institute of Health and Clinical Excellence guidelines Computerized Tomography is considered as the gold standard imaging modality in children presenting with focal deficit following head trauma for assessment and management [9].
MRI fails to demonstrate mineralization on both gradient recalled echo (GRE) sequence and MR angiography (MRA) although it is the investigation of choice for Cerebral Vascular Accident workup. MDCT can easily demonstrate vascular mineralization, which is not possible on conventional CT with 5mm thin axial sections [10].
The mineralization of lenticulostriate arteries supplying to basal ganglia make them more rigid and vulnerable to injuries compared to normal vessels which is more pronounced in childhood as a predisposing factor for Mineralizing Angiopathy following minor trauma [11].
Through this case report, we would like to emphasize on the association of mineralizing angiopathy with basal ganglia infarct after a mild trauma that may lead to gross but often reversible motor symptoms with good neurodevelopmental outcome.

Conclusion

Mineralizing angiopathy is a distinct clinic-radiological entity causing basal ganglia infarct in infants following minor trauma. This case report describes it in a 2 year old child with left sided hemiparesis following minor trauma and recovered well with antiplatelates therapy.
It represents neurological symptoms to persistent lenticulostriate vasculopathy. MDCT is gold standard to demonstrate linear vascular calcifications. Infants with unilateral stroke have a better prognosis than those with bilateral/recurrent stroke and may lead to significant long-term disability.

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